of 0.38% was observed among Th1 and isolates Ku10 and To76 (accession no. AYO83505 and AYO83504, respectively), which belong to *B. valaisiana* genomic group and were isolated from *ricinus* in Sweden (6). A genetic difference of 0.77% was observed between Th1 and *B. valaisiana* strain Tr29 (accession no. ABO91805) isolated from *I. ricinus* in Turkey (7), while the genetic difference between Th1 and *B. burgdorferi* (X15661) was much greater, 6.83%.

This report is the first of genetic detection of *B. valaisiana* in CSF, which indicates a probable association of this genospecies with disease in humans. *B. valaisiana* has been isolated from *I. ricinus* ticks collected from vegetation and from ticks engorged on birds, in several European countries, including Turkey (7). The pathogenic capabilities of *B. valaisiana* are still uncertain; it has been detected by PCR and restriction fragment length polymorphism analysis in skin biopsy specimens from two erythema migrans patients and from patients with mixed infection (erythema migrans and acrodermatitis chronica atrophicans) (4). Indirect evidence suggests that *B. valaisiana* is involved in some chronic clinical manifestations (8).

Borreliosis is difficult to diagnose by serologic evaluation and Western blot interpretation. In our patient, no intrathecal antibodies were produced to support clinical suspicion of disease. The low antibody titers could be attributed to antigenic variation between *B. valaisiana* and *B. burgdorferi sensu stricto*, which was used as antigen because no commercial kit is specific for *B. valaisiana*. Differences between the strain causing infection and the antigen may play a role in the false-negative results (9). The low antibody response in our patient could be caused by antimicrobial drugs and corticosteroid medication.

The high homology of the nucleotide sequence from our patient and respective *B. valaisiana* sequences from other European countries suggests that he likely was infected in Greece. The status of Lyme disease in southern Africa is unknown, but *Ixodes* spp. ticks have been found there, and preliminary evidence indicates that the disease may occur in humans in South Africa (10).

We detected *B. valaisiana* DNA in CSF of a patient with slow progressive spastic paraparesis, which suggests that this microorganism might be the causative agent of the disease. Nucleotide sequence information of *Borrelia* strains from clinical cases and ticks from different countries will elucidate the molecular epidemiology of the disease.

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Baylisascaris procyonis in California

To the Editor: We read with interest the article of Roussere et al. on the distribution of *Baylisascaris procyonis* eggs in northern California communities (1). The widespread dissemination and high density of raccoon latrines in residential areas clearly

pose potential health risks, particularly to young children.

While California has reported more cases of baylisascariasis than any other state, few published studies have reported on the distribution and prevalence of this helminth in the region. In 2001, we conducted a study to determine the presence of *B. procyonis* in the Santa Barbara area by examining roadkill raccoons recovered by animal control staff and stored in a refrigerated facility. On examination, the digestive tract from the stomach to the rectum was removed and tested for *B. procyonis* worms and eggs. Of 26 raccoons examined, 24 (92%, 95% confidence interval 75%–99%) were positive for *B. procyonis* infection. *B. procyonis* worms were found in 85% of the animals examined and eggs were found in 73%. Pet food was frequently found (43%) in the stomach contents of examined raccoons, indicating that such food was made accessible to these animals, either intentionally or inadvertently by residents.

B. procyonis has been identified along the central coast of California, which expands the known range of this helminthic zoonotic agent. This finding, coupled with other published studies, indicates that *Baylisascaris* may be prevalent throughout the state (1,2). Although our study was based on a small sample of selected raccoons, the high infection rate is cause for concern and indicates the potential for human exposure. A presumptive case of *B. procyonis* infection in an 11-month-old child was reported in Santa Barbara in 2003 (1).

Determining the distribution and prevalence of *B. procyonis* is necessary to inform local healthcare providers, public health authorities, and the public of the potential risk. Using road-kill raccoons is a relatively easy method for quickly assessing the presence of *B. procyonis* in a community. Also, this approach avoids trapping and handling live animals and allows stomach contents to be examined to determine where raccoons are feeding. Data from such assessments must be interpreted with caution, since they may not represent all raccoons in an area.

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Streptococcus iniae Discitis in Singapore

To the Editor: *Streptococcus iniae* is a well-recognized fish pathogen that can cause meningoencephalitis in tilapia and trout (1) and necrotizing myositis in red drum (2). We describe the first known human case of *S. iniae* infection in Singapore. This is the second report of spinal infection with this bacterium; however, commercial kits may misidentify *S. iniae*.

The first cases of *S. iniae* infection in humans were reported in Toronto, Canada, in 1995–1996 and included eight patients with bacteremic hand cellulitis and one patient with endocarditis, meningitis, and arthritis (3). Two additional cases were discovered retrospectively, a patient in Ottawa, Canada, with septic arthritis of the knee and a patient from Texas with bacteremic cellulitis. At least two more strains have been isolated from patients in Vancouver, Canada (4). Recently, Lau et al. described two cases of infection in Hong Kong. The first patient had bacteremic cellulitis; the second is recognized as the first patient with *S. iniae* osteomyelitis of the spine (5).

A 73-year-old female Chinese healthcare worker was admitted on October 5, 2003, to Singapore General Hospital. Her symptoms were fever for 3 days before admission and lower back pain that had progressively worsened for the past 2 months, causing her to become bedridden. She was ambulatory before the back pain started and had no history of a fall or injury to the back.

Upon examination, the patient's temperature was 37.1°C. She did not appear septicemic and was hemodynamically stable. No evidence of cellulitis was found, and neurologic examination of the upper limbs showed no abnormalities. Movement and strength of both lower limbs were limited by pain. Reflexes and plantar responses were normal, and no focal tenderness over the spine was found; chest x-ray results were normal. Laboratory tests showed the following: leukocytes 12.91 x 10^9/L, hemoglobin 9.9 g/dL, platelets 261 x 10^9/L, serum albumin 20 g/L, bilirubin 17 μmol/L, alkaline phosphatase 132 U/L, alanine transaminase 16 U/L, and aspartate transaminase 23 U/L. Renal function tests were within normal limits. The erythrocyte sedimentation rate was 115 mm/h, and C-reactive protein was 88.4 mg/L. No bacteria were grown from blood cultures. Treatment with empiric intravenous cefazolin was started.